Bacteremia Caused by Vibrio hollisae

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Vibrio hollisae was recovered from the bloodstream of a 36-year-old man with chronic active hepatitis who was admitted to the hospital with signs of gastrointestinal illness. V. hollisae is an infrequent human pathogen associated with seafood ingestion. No etiologic link to seafood was demonstrated in this case report.

The first report of pathogenicity of Vibrio hollisae was its coincident recovery with Cryptococcus neoformans from the bloodstream of a comatose man suffering from cirrhosis, hepatic encephalopathy, and pneumonia (10). The second report described the recovery of the microorganism from the bloodstream of a man who ate fried catfish. This man had a past medical history of cirrhosis and splenectomy (7). We present a case of bacteremia caused by V. hollisae in a 36-year-old male with chronic active hepatitis, portal hypertension, and esophageal varices with gastrointestinal bleeding.

Case report. The patient was admitted to the hospital complaining of abdominal pain and diarrhea for 2 days. He experienced cramps, multiple episodes of diarrhea, nausea, vomiting, intermittent chills, fever, and fatigue. His temperature was 100°F (ca. 37.8°C) (rectal). His leukocyte count was 12,100/mm², with 76% segmented neutrophils and 18% band neutrophils. Stool was cultured for Salmonella spp., Shigella spp., and Campylobacter spp., examined for ova and parasites, and assayed for Clostridium difficile toxin. Blood was drawn for culture. All of the test results were negative. The patient was hydrated with intravenous fluids. His abdominal pain and diarrhea resolved. The patient was discharged from the hospital with a therapeutic regimen of doxycycline (Colace), trimethoprim-sulfamethoxazole, and ferrous sulfate.

The patient was readmitted 6 days later complaining of abdominal pain, vomiting, and diarrhea. The admission diagnosis was acute dehydration secondary to improper self-dosing of his medications. He had no fever or chills at the time of admission, but within 24 h, his temperature rose to 101.4°F (ca. 38.6°C), accompanied by vertigo. Stool samples were obtained for bacterial culture and ova and parasite examination as described above. The test results were negative. Two blood samples were drawn for culture. The patient was administered cefoxitin sodium and gentamicin sulfate empirically. After 24 h of incubation at 35°C, the aerobic blood culture bottle (BACTEC 6B; Johnston Laboratories, Inc., Towson, Md.) contained a gram-negative coccobacillus and the anaerobic blood culture bottle (BACTEC 7D) revealed a large gram-negative rod. In vitro disk diffusion susceptibility studies (2) revealed identical susceptibility patterns for the microorganisms to all of the antimicrobial agents tested. The patient's therapy was changed to ampicillin. The patient defervesced, the abdominal pain disappeared, and the diarrhea cleared. The patient was discharged after 6 days in the hospital.

The bacteremia was monomicrobial. The growth charac-

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MICs and MBCs (Sceptor System; Johnston Laboratories) were as follows (in micrograms per milliliter): ampicillin, <0.5; ticarcillin, <4; piperacillin, <4; cephalothin, <1; cefotaxime, <1; amikacin, <1; gentamicin, <0.25; tobramycin, <0.25; trimethoprim-sulfamethoxazole, <2/38; and chloramphenicol, <2.

The biochemical test results were oxidase positive, spot indole positive, motility positive, and Uni-OF Glucose tube (Flow Laboratories, Inc., McLean, Va.) positive for fermentation; and TSI agar (BBL Microbiology Systems) indicated an alkaline slant over a strong acid butt. The API 20E strip revealed intense indole production. The glucose and arabinose reactions were imprecise owing to bright lime color production instead of yellow (positive) or blue to blue-green (negative). Carbohydrate fermentation tests were negative in both bottles (GIBCO, Lawrence, Mass.) with arabinoxyl, xylose, lactose, sucrose, maltose, and mannitol. Nitrate reduction to nitrite was positive. We repeated all of the test results. The final identification was made by the State of Connecticut Department of Health Services and rechecked by the Enteric Identification Laboratory of the Centers for Disease Control.

Discussion. The only problem encountered in identifying the microorganism was the negative glucose reaction observed with the API 20E system compared with the positive reactions of the TSI slant and Uni-OF Glucose tube. The
API profile number reflects the negative glucose reaction. This reaction may be acceptable in the API system, but it conflicts with other accounts (5) of the ability of the microorganism to produce acid from glucose.

Enteric illness caused by *Vibrio* spp. (3–7, 10) is associated with the consumption of seafood. To investigate the bacterial sources of the infection, the patient was interviewed about his diet during a follow-up examination. The interview was hindered by language barriers. We learned that the patient, a Laotian, purchased a dried and salted fish at a local Southeast Asian food store. He was unable to describe the fish but referred to it in Laotian with a word meaning “bone.” We could not determine the brand. Normally he cooked the fish prior to eating it. He indicated that he ate the fish uncooked during a lengthy community-wide power outage. The incubation period prior to the onset of his symptoms was indeterminable. This was the only fish or seafood connection we could make. One of us (E.L.R.) visited the store and purchased representative packages of three different brands of dried and salted fish. Each product was analyzed in accordance with an accepted food-sampling method (8). The suspensions were subcultured on TSA II and Mueller-Hinton II agar media because the microorganism grew well on these media during the initial isolation and identification procedures. The plates were incubated at 35°C in 5% CO₂. Each bacterial colony was subjected to an oxidase test and a Gram stain. We were unable to recover *V. hollisae* from these samples.

*Vibrio* spp. are not sought in our pathogenic stool culture examination, and thiosulfate-citrate-bile salts-sucrose agar is not routinely used. It is unfortunate that we were unable to relate the presentation of diarrhea to the subsequent bacteremia. Recent evidence suggests that *V. hollisae* may share with *Vibrio vulnificus* a predilection for bloodstream invasion in people with liver abnormalities (3, 11). The availability of iron may play a role in pathogenesis. *Yersinia enterocolitica* septicaemia was attributed to an overdose of ferrous sulfate (9). Pathogenic vibrios can produce siderophores to bind iron for growth (1). *V. vulnificus* was unable to grow in normal human serum, but the addition of ferric ammonium citrate reduced a 50% lethal dose of 10⁶ to 1.1 cells in a mouse model of mortality (12). Our patient received ferrous sulfate as a supplement for chronic anemia, and improper self-dosing of his medication may have exacerbated the underlying disease process.

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### LITERATURE CITED


